ment conditions.

KING POLICY

Restricted Filtration [RF]	Restricted No Treat. [RNT]	Restricted Workstation [RWS]
0.4	0.6	0.1
(0.1-2.1)	(0.1-3.3)	(0.0-0.5)
674	634	573
(575-791)	(533-755)	(488-671)
0.02	0.05	0.01
(0.013-0.047)	(0.025-0.103)	(0.008-0.028)
2 : 14.02	5.3 ± 15.32	2.6 ± 14.0
114.> ± 22.9	19.8 ± 25.1	10.2 ± 22.9
20.8 ± 2.4	3.8 ± 2.6	2.4 ± 2.4
23.9 ± 0.4	23.7 ± 0.4	23.6 ± 0.4
33.4 ± 3.5	28.3 ± 3.9	35.9 ± 3.5
507.9 ± 62.4	586.4 ± 67.8	564.9 ± 62.4

for nonsmoking and smoking areas of

)KING POLICY

RF	RNT	RNT
Smoking	Nonsmoking	Smoking
1.2	0.4	1.0
(0.7-2.1)	(0.2-0.8)	(0.4-2.2)
676	597	698
(550-644)	(550- 647)	(624-781)
0.04	0.05	0.06
:2) (0.030-0.049)	(0.038-0.063)	(0.039-0.079)
126.1 ± 8.8	19.3 ± 9.0	35.6 ± 12.5
236.6 ± 20.0	9.3 ± 20.5	35.6 ± 28.6
4 ± 3.6	0.3 ± 3.8	10.3 ± 5.2
± 0.2	23.7 ± 0.2	23.8 ± 0.2
33.6 ± 1.1	28.2 ± 1.2	28.7 ± 1.6
503.6 ± 42.0	648.8 ± 43.0	474.5 ± 60.3

ing areas

MULTIPLE MEASURES OF PERSONAL ETS EXPOSURE IN A POPULATION-BASED SURVEY OF NONSMOKING WOMEN IN COLUMBUS, OHIO

M. W. Ogden, R. A. Davis, K. C. Maiolo, M. F. Stiles, D. L. Heavner, R. B. Hege, and W. T. Morgan

R. J. Reynolds Tobacco Co., Research & Development, Winston-Salem, North Carolina, USA

ABSTRACT

Environmental tobacco smoke (ETS) exposure was assessed continuously over a one-week period in a group of 96 nonsmoking, married women in Columbus, Ohio. Markers used to estimate ETS exposure were airborne nicotine and 3-ethenylpyridine (3-EP), and salivary cotinine. For purposes of assessing relative ETS exposure, the women were categorized according to husband's smoking status (*e.g.*, ETS-exposed if husband smokes). The relative exposures measured were: 8.6 for cotinine; 13.4 for nicotine; and 22.0 for 3-EP. These relative exposures are significantly larger than those assumed in recent ETS risk assessments and tend to question the validity of risk estimate corrections for so-called "background exposure." The same ratios measured for employed subjects indicate that workplace exposure is 10-fold lower than home exposure due to living with a smoker. In addition, these data also suggest that cotinine alone may be an inadequate marker for estimating ETS exposure in the general population. Median concentrations (all women) were found to be: 0.15 $\mu g/m^3$ (3-EP); 0.43 $\mu g/m^3$ (nicotine); and 0.8 ng/mL (salivary cotinine).

INTRODUCTION

An important issue in ETS risk assessment is that of background correction. As far as possible, epidemiologic studies employ control groups which are not exposed to the agent or action under investigation. Although tobacco use in the United States and the world is prevalent, and the potential exists for widespread exposure to ETS, some have claimed that ETS is ubiquitous (1,2) and make adjustments to risk estimates for exposure in control groups. Little is known about the magnitude of relative exposure in the population at large, and an upward adjustment to any risk estimate seems premature in light of the available evidence. In a recent risk assessment (2), the U.S. EPA adjusted the pooled U.S. risk estimate for a background correction factor (relative exposure) of 1.75. This factor is derived solely from relatively few measurements of cotinine. The generalizability to the U.S. population of much of these data is dubious, owing to the fact that virtually all of the studies cited were either of non-U.S. subjects or of a highly specialized subset of U.S. subjects (*e.g.*, only Hispanics, clinic patients and professional staff, etc.). Added to this is the observation that indices of exposure to ETS based on biologic markers, air monitoring, and questionnaires are not tightly correlated, and further, that single measures of ETS components or biologic markers are inadequate for characterizing normal exposure (3). In an attempt to partially rectify this lack of relevant data, the current study was designed. As such, this study has the unique distinction of;

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(1) being representative of the U.S. female population with subject's ETS exposure status categorized exactly in accordance with the relevant epidemiology, (2) employing multiple markers of ETS exposure, and (3) measuring each marker over an extended time period.

SUBJECTS AND METHODS

Subject Selection and Study Design. Self-reported nonsmoking, married females (n=105) were recruited at random by a market research agency in Columbus, Ohio. Columbus was chosen as the test site due to its status as "one of the most average cities in the country" with its residents comprising a "demographically normal" segment of the U.S. population (4). A "nonsmoker" was defined as one who did not currently smoke and had not done so for at least six months prior to the study. A "never-smoker" was defined as one who had not ever smoked on a regular basis. Subjects were selected to fill cells defined by combinations of age, income, employment status, and husband's smoking status. The primary criteria were subject's employment status and husband's smoking status, for which cells were equally sized. For practical considerations, cells based on the remaining secondary criteria were only approximately equally sized. Subjects were considered employed if they worked full-time (35+ hours/week) outside the home and unemployed if they did not work at all outside the home. None of the subjects worked in a location with a smoking ban. Subjects were monitored continuously for ETS exposure (airborne nicotine and 3-ethenylpyridine) and nicotine intake (salivary cotinine) during one week (February, 1991) as they went about their normal daily routines. In addition, subjects married to smokers monitored airborne 3-EP and nicotine levels in their homes continuously for the week. Both pre- and post-sampling questionnaires were administered by trained interviewers at the agency's facility. Extensive data were gathered regarding dietary and lifestyle habits, subject whereabouts, and exposure to ETS. In addition, each subject maintained a daily activity diary during the study recording the same information on an hourly basis (excluding diet). Only the objective estimates of ETS exposure (nicotine, 3-ethenylpyridine, and cotinine), along with questionnaire responses regarding subject smoking history, are reported here.

Nicotine and 3-Ethenylpyridine Determination. All subjects wore a personal, diffusion-based monitor continuously for five days except during sleep, when monitors were placed at bedside. In addition, wives of smoking husbands placed two monitors at fixed locations in their homes; one in a "heavy use" area and one in a "light use" area. Heavy and light use areas were targeted to be rooms where the husband did most of his in-home smoking and little, if any, smoking, respectively. Locations typically used by the participants were a den or living room for the heavy use area and an unused bedroom or a child's bedroom for the light use area. The monitors collected nicotine and 3-EP (a nicotine combustion product) on a sodium bisulfate-treated filter which was then extracted and analyzed by gas chromatography with *N*-thermionic detection (5). With an exposure duration of five days, limits of detection (LOD) for 3-EP and nicotine were 0.04 and 0.13 $\mu g/m^3$, respectively.

Cotinine Determination. Saliva samples were obtained by having the subject vigorously chew a Salivette (Sarstedt, Inc., Newton, NC) for 1 min. Four samples were obtained per subject, approximately one every other day, over a period of seven days (the first saliva sample was taken one to two days prior to the start of air monitoring). Salivettes were frozen immediately after sampling, transferred to the laboratory under dry ice, and stored frozen until analyzed. Duplicate analyses for cotinine were performed on each sample by the radioimmunoassay method of Langor 10 ng/mL was used to differentiate smol of saliva in buffer, LOD for cotinine in

RESULTS

Of the 105 subjects recruited for this stu reported ex-smokers) successfully compl (two reported never-smokers and six rep smokers ("deceivers") based on mean sa were excluded from analysis. All four s cotinine in concentrations > 10 ng/mL. cotinine results, were retained. It is lik smokers; however, their data were retai

Misclassifying two out of 70 reported n misclassification rate of 2.9%; misclassi an ex-smoker misclassification rate of 1 reported nonsmokers results in a nonsm

Following deletion of the eight deceiver objective estimates of personal ETS exp presented in Table 1. Data are presente smoking status and subject's employme subject's workplaces. For employed su them while at work.

Table 1. Analytical results (medians) o husband's smoking status and subject's

Subject's husband smokes?	Subject employed?
yes	yes
no	yes
yes	no
no	no
yes	all subjects
no	all subjects
all subjects	yes
all subjects	no
all subjects	all subjects

[a]Median=0; value reported is LOD/2
[b]Median < LOD

Similarly, estimates of home ETS expo Table 2. Data are presented according home and the subject's employment st the ratio of median values in all pertin

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mployment status and husband's smoking
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obtained by having the subject vigorously
for 1 min. Four samples were obtained per
er a period of seven days (the first saliva
start of air monitoring). Salivettes were
to the laboratory under dry ice, and stored
cotinine were performed on each sample by

the radioimmunoassay method of Langone (6). In general, a cut-point concentration of 10 ng/mL was used to differentiate smokers from nonsmokers (7). Using a 1:10 dilution of saliva in buffer, LOD for cotinine in saliva was determined to be 0.18 ng/mL.

RESULTS

Of the 105 subjects recruited for this study, 104 (70 reported never-smokers and 34 reported ex-smokers) successfully completed the sampling phase. Eight of these subjects (two reported never-smokers and six reported ex-smokers) were deemed to be active smokers ("deceivers") based on mean salivary cotinine values > 10 ng/mL, and their data were excluded from analysis. All four samples for each of these subjects contained cotinine in concentrations > 10 ng/mL. Three additional subjects, with borderline cotinine results, were retained. It is likely that these three subjects were occasional smokers; however, their data were retained nonetheless.

Misclassifying two out of 70 reported never-smoking subjects results in a never-smoker misclassification rate of 2.9%; misclassifying six out of 34 reported ex-smokers results in an ex-smoker misclassification rate of 17.6%; and, misclassifying eight out of 104 reported nonsmokers results in a nonsmoker misclassification rate of 7.7%.

Following deletion of the eight deceivers, summary statistics were computed for the objective estimates of personal ETS exposure in the remaining subjects, and the results are presented in Table 1. Data are presented according to all combinations of husband's smoking status and subject's employment status. Smoking was permitted in all of the subject's workplaces. For employed subjects, 37 of 45 (82%) observed smoking near them while at work.

Table 1. Analytical results (medians) of week-long personal monitoring according to husband's smoking status and subject's employment status.

Subject's husband smokes?	Subject employed?	n	3-EP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)	Cotinine (ng/mL)
yes	yes	23	0.66	2.17	2.37
no	yes	22	0.06	0.21	0.40
yes	no	25	0.36	0.87	2.125
no	no	26	0.02[a]	0.08[b]	0.23
yes	all subjects	48	0.44	1.61	2.33
no	all subjects	48	0.02[a]	0.12	0.27
all subjects	yes	45	0.19	0.89	0.93
all subjects	no	51	0.11	0.26	0.64
all subjects	all subjects	96	0.15	0.43	0.82

[a]Median=0; value reported is LOD/2
[b]Median < LOD

Similarly, estimates of home ETS exposure in the smoking households are summarized in Table 2. Data are presented according to all combinations of sampler location within the home and the subject's employment status. Relative exposure estimates were calculated as the ratio of median values in all pertinent categories in Tables 1 and 2, and the results are

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Table 2. Analytical results (medians) of week-long area monitoring in smoker's homes.

Home area*	Subject employed?	n	3-EP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)
light use	yes	23	0.68	1.14
light use	no	25	0.25	0.25
heavy use	yes	23	0.80	3.30
heavy use	no	25	0.62	1.50
avg light & heavy use	yes	23	0.81	2.39
avg light & heavy use	no	25	0.43	0.93
light use	all subjects	48	0.36	0.56
heavy use	all subjects	48	0.63	2.45
avg light & heavy use	all subjects	48	0.48	1.63

*For definitions of light and heavy use, see text.

summarized in Table 3. The ratios shown in row 1 (ratio between "smoking husband" and "nonsmoking husband" categories) employ the definition of exposed and unexposed subjects used in most epidemiology studies and conform to the definitions used by NRC (1) and EPA (2).

Table 3. Relative exposure estimates.

Relative exposure	3-EP	Nicotine	Cotinine
all subjects: S/NS husband	22.0††	13.4††	8.6††
unemployed subjects: S/NS husband	18.0††	10.9††	9.2††
employed subjects: S/NS husband	11.0††	10.3††	5.9††
all subjects: employed/unemployed	1.7*	3.4†	1.5*
subjects w/ NS husbands: employed/unemployed	3.0*	2.6†	1.7*
subjects w/ S husbands: employed/unemployed	1.8*	2.5†	1.1*
heavy home monitors: employed/unemployed	1.3*	2.2*	---
light home monitors: employed/unemployed	2.7†	4.6†	---
avg light & heavy: employed/unemployed	1.9*	2.6†	---
home monitors: heavy/light use areas	1.8*	4.4††	---

*Individual measures of central tendency not significantly different ($p > 0.05$)
†Individual measures of central tendency significantly different ($0.01 \leq p \leq 0.05$)
††Individual measures of central tendency significantly different ($p \leq 0.0003$)

DISCUSSION

Cotinine, a major nicotine metabolite with a half-life in humans of *ca.* 20 h (8), is considered the only viable biochemical marker of ETS exposure, although its use is not without limitations. Body burden of cotinine is presumed to reflect exposure to nicotine; however, exposure to nicotine does not necessarily equate with exposure to ETS. Due to unusual decay characteristics, nicotine lingers in many environments long after other

tobacco smoke constituents have disap
overestimating ETS exposure (9). Th
the diet and large differences in indiv
quantitative application of nicotine an
Ethenylpyridine, a combustion produc
exposure since 3-EP decays at rates si
(11,12).

The concentrations of the airborne ma
exposures are considerably lower than
(13), even for the most heavily expos
This is due primarily to the inclusion
absence of ETS (during sleep, travel,
true exposure. From a population per
virtually all of their ETS exposure cor
concentrations from personal monitori
to the median average home concentra
monitor data from each home, Table 2

The relative exposure data in Table 3
magnitude of exposure ratios between
husband" category (Table 3, row 1) su
(*e.g.*, background correction factor "Z
The data reported here from three mar
demographically representative cross-s
are at least five- to ten-times greater
subjects (Table 3, row 3) indicate that
approximately 10-fold lower than hom
presumed by many that the only major
with a smoker is the workplace (2,14,
workplace exposure appears to be an o
exposure. Such data contradict recent
comparable with home ETS levels, anc
biomarkers demonstrate that nonspousa
from spousal smoking" (2, p. 1-12). A
here is that interpretation of workplace
questionnaire data, 24-hour exposure m
spurious conclusions. Such measures c
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same trend is noted for 3-EP monitor d
significant). A cursory analysis might
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We note that just such a conclusion wa
However, analysis of the relative magn
categories (Table 3, rows 7-9) demonst
employed wives of smokers. Conseque
and is, instead, a confounded measure
observed in-home smoking behavior of

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g area monitoring in smoker's homes.

n	3-EP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)
23	0.68	1.14
25	0.25	0.25
23	0.80	3.30
25	0.62	1.50
23	0.81	2.39
25	0.43	0.93
[illegible]	0.36	0.56
[illegible]	0.63	2.45
48	0.48	1.63

l (ratio between "smoking husband" definition of exposed and unexposed nform to the definitions used by NRC

	3-EP	Nicotine	Cotinine
	22.0††	13.4††	8.6††
	18.0††	10.9††	9.2††
	11.0††	10.3††	5.9††
	1.7*	3.4†	1.5*
d	3.0*	2.6†	1.7*
	1.8*	2.5†	1.1*
	1.3*	2.2*	---
	2.7†	4.6†	---
	1.9*	2.6†	---
	1.8*	4.4††	---

erent ($p > 0.05$)
t ($0.01 \leq p \leq 0.05$)
nt ($p \leq 0.0003$)

e in humans of *ca.* 20 h (8), is TS exposure, although its use is not sumed to reflect exposure to nicotine; equate with exposure to ETS. Due to ny environments long after other

tobacco smoke constituents have disappeared, resulting, in many instances, in nicotine overestimating ETS exposure (9). This, along with possible consumption of nicotine in the diet and large differences in individual rates of metabolic clearance (10), limits the quantitative application of nicotine and cotinine in exposure assessment at low doses. 3-Ethenylpyridine, a combustion product of nicotine, is a more objective estimate of ETS exposure since 3-EP decays at rates similar to other ETS components (including particles) (11,12).

The concentrations of the airborne markers (Table 1) indicate that long-term personal exposures are considerably lower than predicted from short-term area monitoring data (13), even for the most heavily exposed group (*i.e.*, nonsmokers married to smokers). This is due primarily to the inclusion of significant portions of sampling time in the absence of ETS (during sleep, travel, etc.) and is, of course, a more accurate reflection of true exposure. From a population perspective, it appears that for wives of smokers, virtually all of their ETS exposure comes from the home. The 3-EP and nicotine median concentrations from personal monitoring (Table 1, rows 1 and 3) are essentially equivalent to the median average home concentrations (derived from averaging the light and heavy monitor data from each home, Table 2, rows 5 and 6).

The relative exposure data in Table 3 support several important conclusions. First, the magnitude of exposure ratios between the "smoking husband" and the "nonsmoking husband" category (Table 3, row 1) suggests that factors used in recent risk assessments (*e.g.*, background correction factor "Z" = 1.75 (2)) are in error for the U.S. population. The data reported here from three markers measured over a fairly long time period in a demographically representative cross-section of U.S. women suggest true values of "Z" are at least five- to ten-times greater. Second, the same ratios measured for employed subjects (Table 3, row 3) indicate that workplace exposure due to working with smokers is approximately 10-fold lower than home exposure due to living with a smoker. It is presumed by many that the only major source of ETS exposure for a nonsmoker not living with a smoker is the workplace (2,14,15). While this may be true, the extent of workplace exposure appears to be an order of magnitude lower than "spousal smoking" exposure. Such data contradict recent opinion that "[w]orkplace ETS levels are generally comparable with home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that nonspousal exposures to ETS are often greater than exposure from spousal smoking" (2, p. 1-12). A third important conclusion from the data reported here is that interpretation of workplace exposure based upon nondiscriminating data (*e.g.*, questionnaire data, 24-hour exposure monitoring, cotinine measures, etc.) may lead to spurious conclusions. Such measures cannot discriminate among home, workplace and other exposures in the way that discreet air monitoring in specific locations can. For instance, Table 3 (row 6) indicates a 2.5-fold increase in the week-long nicotine monitor data for employed wives of smokers compared with unemployed wives of smokers (the same trend is noted for 3-EP monitor data; however, the difference is not statistically significant). A cursory analysis might conclude that employed wives of smokers have significant additional exposure from the workplace over their unemployed counterparts. We note that just such a conclusion was reached recently (15) based on questionnaire data. However, analysis of the relative magnitude of home exposure between these two categories (Table 3, rows 7-9) demonstrates that home exposures are two-fold higher for employed wives of smokers. Consequently, the apparent workplace effect is misleading and is, instead, a confounded measure of home exposure. There is no difference in the observed in-home smoking behavior of husbands between the two categories; however,

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homes in the employed subject category were significantly smaller than homes in the unemployed subject category. Thus, we conclude that the increased home exposure is due to the smaller home size, a potential socioeconomic confounder. When the week-long personal monitoring data are corrected for exposure in the home (reported time at home combined with average home monitor data), the apparent workplace effect disappears. Also of interest in this same category are the virtually identical median levels of salivary cotinine (compared to a significant 2.5-fold difference in nicotine exposure and an insignificant 1.8-fold difference in 3-EP exposure). This suggests that of the three markers used in this study to characterize ETS exposure, cotinine is the least discriminating. In conjunction with additional problems outlined previously, and the noted overall trend in differentiating ability (Table 3, row 1) among the markers, cotinine appears to significantly underestimate true differences in ETS exposure.

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HOW MUCH DOES ENVIRO CONTRIBUTE TO THE BUI

A. Raynal[1], P.S. Burge[2], A. Robertso

[1]Occupational Health Department, Bri
[2]Occupational Lung Disease Unit, Eas
[3]Institute of Occupational Health, Birr
[4]Imperial Cancer Research Fund, Heal
Maudsley Hospital, London, UK
[5]British Gas (West Midlands), Solihul
[6]British Gas (West Midlands), Solihul

ABSTRACT

A longitudinal cohort study was condu
Index (BSI) is attributable to exposure
smokers.

A cross-sectional analysis of the initia
smoking status could be validated thro
BSI and perceived ETS exposure were
levels adjacent to smokers were signif
found between the Personal Symptom

It is concluded that smoking status an
on the BSI than objectively measured

INTRODUCTION

In a study of 4,373 office workers, Ro
building sickness were more common
smoke at work than those who were n
workers (2) found that symptoms were
Environmental Tobacco Smoke (ETS).
biological measurements of ETS were
validate their exposures.

As many employers are taking the init
is useful for those concerned to have i
usually involuntary) exposure to ETS
the workplace. The BSI is the mean n
building syndrome per occupant. If thi
would be an additional justification for

The regional headquarters of British G
Solihull, accommodates approximately

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